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Blood Lactate
Implications for Training and Sports Performance

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Summary

The blood lactate response to exercise has interested physiologists for over fifty years, but has more recently become as routine a variable to measure in many exercise laboratories as is heart rate. This rising popularity is probably due to: a) the ease of sampling and improved accuracy afforded by recently developed micro-assay methods and/or automated lactate analysers; and b) the predictive and evaluative power associated with the lactate response to exercise. Several studies suggest that the strong relationship between exercise performance and lactate-related variables can be attributed to a reflection by lactate during exercise of not only the functional capacity of the central circulatory apparati to transport oxygen to exercising muscles, but also the peripheral capacity of the musculature to utilise this oxygen. For example, several studies contrast the relationship between $\dot{V}O_{2\,max}$ and endurance running performance with that between a lactate variable and the same running performance. In every study, the lactate variable is more highly correlated with performance. Similarly, prescribing training intensity as a function of the lactate concentration elicited by the training may prove to be a means of obtaining a more homogeneous adaptation to training in a group of athletes or subjects than is obtained by setting intensity as a function of maximal heart rate or % $\dot{V}O_{2\,max}$. A review of the recent literature shows that the lactate response to supramaximal exercise is a sensitive indicator of adaptation to 'sprint training' and is correlated with supramaximal exercise performance. This review also describes the possible applications of lactate measurements to enhance the rate of recovery from high intensity exercise. Although the lactate response to exercise is reproducible under standardised conditions it can be influenced by the site of blood sampling, ambient temperature, changes in the body's acid-base balance prior to exercise, prior exercise, dietary manipulations, or pharmacological interpretation.

The attention increasingly being rendered to blood lactate in exercise physiology literature should not be interpreted as meaning that similar experiments were not performed previously. Jervell (1929) described the curvilinear relationship of lactate to power output during exercise over 50 years ago. This same work was one of the earliest to observe a more rapid decrease in circulating lactate after exhaustive exercise if an individual exercised during recovery than if he/she sat passively. Exercise physiologists are still intrigued with a 'critical metabolic level below which there (is) no increase in blood lactate as a result of exercise although above this level such an increase (does) occur.' This quotation, from Owles (1930), is the first description of what has been more recently termed the 'anaerobic threshold' (Wasserman et al. 1973), which is hardly a recently discovered phe-

nomenon. The longitudinal effect of endurance training on the lactate response to exercise was described 50 years ago (Bang 1936). Bang also speculated on the rate and source of lactate production and its fate during exercise, questions which are still pertinent for scientific investigation and debate (see Donovan & Brooks 1983).

This review will be limited to discussing how the measurement of blood lactate concentration during and/or after exercise, and the calculation of a related variable, can and is being used in the exercise laboratory and in the field. This includes how lactate is used: (a) to evaluate performance of supramaximal or endurance exercise; (b) to predict performance; and (c) its use as a training tool. Most of the content of this review is based on published references in English (with a few exceptions) and some of my own previously unpublished observations. Unless stated otherwise, references to lactate concentration refer to blood or plasma concentrations.

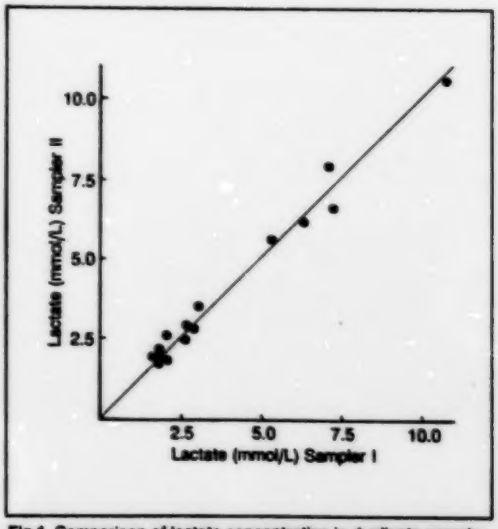

Fig 1. Comparison of lactate concentration in duplicate samples obtained from exercising subjects. Two different individuals, an experienced sampler (sampler I) and a novice (sampler II), obtained one sample each from the same subject. [Reproduced with permission from Karlsson et al. 1983b.]

Table I. Some examples of automated and semi-automated lactate analysers which are commercially available

Model	Method	Blood sample volume (μL)
Yellow Springs Instruments model 23L	Enzyme electrode	25
Kontron Medical ('Roche') model 640	Enzyme electrode	25
Alpha Laboratories 'Analox' LM2	Enzyme electrode	5
'Bifok' FIA 05	Enzyme fluorometric Flow injection analysis	25

1. Blood Sampling and Lactate Analysis

The number of publications appearing annually concerning lactate and exercise in humans has probably increased exponentially in the last decade. A major contributing factor to the popularity of lactate measurements is the ease of sampling and improved accuracy afforded by micro-assay methods (Karlsson et al. 1983b; Maughan 1982). Microblood sampling is relatively innocuous for the subject; and only a few drops of blood are required and can be readily obtained from a punctured fingertip or earlobe. The procedure is no longer a difficult one which requires hundreds of practice samplings before one can be considered a reliable sampler. This is demonstrated in figure 1, which compares the assayed lactate concentration in duplicate samples obtained from the punctured fingertip of an exercising subject. Sampler I was an experienced sampler with over 5 years of sampling experience, compared with sampler II who received 10 minutes of instruction and was permitted 2 practice punctures before taking the plotted samples and expelling the contents of a 20μL pipette into deproteinising acid. This ease of accurate sampling, in addition to the recent proliferation of automatic or semi-automatic lactate analysers (table I), has undoubtedly contributed to the increasing popularity of measuring lactate as a routine

variable to be considered in any exercise physiology experiment.

2. *Endurance Exercise*

A myriad of studies have demonstrated via both cross-sectional and longitudinal comparisons that endurance training results in decreased muscle and blood lactate accumulation at given absolute or relative exercise intensities (Bang 1936; Ekblom et al. 1968; Karlsson et al. 1972). An applied extension of such observations has been the predictive power associated with the lactate response to exercise. Lactate-related variables account for a larger proportion of the variation in endurance exercise performance than other variables traditionally determined in the exercise laboratory – including the measurement of maximal aerobic power ($\dot{V}O_{2max}$). Table II illustrates a few of the studies which contrast the relationship of $\dot{V}O_{2\,max}$ and running performance with the relationship of a lactate variable and the same running performance. In every case, the lactate variable is more highly correlated with running performance.

Table II. Comparison of the correlations between competitive running/walking performance and $\dot{V}O_{2max}$ with the correlations between performance and a calculated variable based on the blood lactate response to submaximal treadmill exercise

Reference	Performance (km)	Correlation	
		$\dot{V}O_{2max}$	lactate variable
Farrell et al. (1979)	42.2 (run)	0.91	0.98
	19.3	0.91	0.97
	15.0	0.89	0.97
	9.7	0.86	0.96
	3.2	0.83	0.91
Jooste et al. (1981)	90 (run)	$p > 0.05$	0.93
Kumagai et al. (1982)	5 (run)	0.46	0.95
	10	0.75	0.84
	6.2	0.486	0.835
Hagberg & Coyle (1983)	Racewalking	0.62	0.94
Lehmann et al. (1983)	30 (run)	0.71	0.76
Williams & Nute (1983)	Half-marathon	0.81	0.88
Allen et al. (1985)	10 (run)	< for lactate	> 0.89

2.1 Lactate Variables

The aerobic threshold (Kindermann et al. 1979), anaerobic threshold (Wasserman et al. 1973), aerobic-anaerobic threshold (Mader et al. 1976), onset of blood lactate accumulation (OBLA) [Sjödin & Jacobs 1981], onset of plasma lactate accumulation (Farrell et al. 1979), lactate threshold (Ivy et al. 1980), lactate turning-point (Davis et al. 1983), maximal steady-state (LaFontaine et al. 1981), the individual anaerobic threshold (Stegmann et al. 1981), excess lactate (Williams et al. 1967), aerobic capacity (Davies et al. 1970) are all terms which have been used to describe calculated variables related to the kinetics of lactate accumulation during steady-state submaximal exercise. Some of these terms refer to the exercise intensity at which a given lactate accumulation occurs; others refer to the intensity which elicits a lactate concentration just above resting values. These intensities can be expressed in absolute terms as treadmill running velocity, cycle ergometer power outputs, or oxygen uptake. They can also be expressed as the percentage of $\dot{V}O_{2\,max}$ at which they occur. Both expressions are increased with endurance training (Ekblom et al. 1968; Hermansen & Stensvold 1972). Similar to the manner in which various types of athletes have been characterised by their $\dot{V}O_{2\,max}$ (Bergh et al. 1978), table III lists the exercise intensity which elicits 4 mmol/L lactate during steady-state exercise for several athlete groups. German exercise physiology groups, in particular, have described the lactate response to exercise of various athlete and age groups for both sexes (Heck et al. 1983; Liesen et al. 1979).

2.2 Multiple Blood Samples Not Required

The kinetics of lactate accumulation dictate that a subject who accumulates 2 to 3 mmol/L lactate

Table III. Exercise intensity which elicits 4 mmol/L blood lactate during steady-state exercise in various competitive athletic groups

Group	Intensity	Exercise test	Reference
Male kayakers	220W	Arm cranking	Tesch and Lindeberg (1984)
Female kayakers	137W		
Male weight/power lifters	100W		
Male body builders	100W		
Male distance runners	5.15 m/sec	Treadmill, 1.4% grade	Hess et al. (1983)
Male middle distance runners	4.78 m/sec		
Male 400m runners	4.02 m/sec		
Male 400m runners	4.50 m/sec	Treadmill, flat	Svedenhag and Sjödin (1984)
Male 800m runners	4.90 m/sec		
Female 800m runners	3.99 m/sec	Treadmill, 1.4% grade	Föhrenbach et al. (1981)
Male 1500m runners	5.2 m/sec	Treadmill, flat	Svedenhag and Sjödin (1984)
Female 1500m runners	4.26 m/sec	Treadmill, 1.4% grade	Föhrenbach et al. (1981)
Male 5000m runners	5.6 m/sec	Treadmill, flat	Svedenhag and Sjödin (1984)
Male marathoners	5.5m/sec		
Male ultramarathoner	5.5 m/sec	Treadmill, flat	Jacobs et al. (1980)
Male soccer players	4.15 m/ssc	Treadmill, 1.5% grade	Hollmann et al. (1981a)
Male modern pentathletes	4.7 m/sec	Treadmill, 1.4% grade	Hess et al. (1983)
Male ice hockey players	3.72 m/sec		
Male field hockey players	4.21 m/sec		
Female field hockey players	3.40 m/sec		
Male rowers	292W	Cycle	
Male rowers	340W	Cycle	Roth et al. (1981)
Male professional cyclists	317W	Cycle	Hess et al. (1983)
Male professional cyclists	390W	Cycle	Roth et al. (1981)
Male swimmers	1.35 m/sec	Crawl swimming	Olbrecht et al. (1985)

at a relatively low exercise intensity will also have a lower exercise intensity at 4 to 5 mmol/L when compared with a fitter subject (Tesch et al. 1981). In other words, the results obtained by the various methods used to calculate these variables must be highly correlated. Those same characteristics which contribute to the delay of lactate accumulation during progressive exercise also contribute to a lower lactate accumulation after an acute bout of exercise for 4 to 7 minutes (Jacobs et al. 1983a). Therefore, it seems quite justifiable to use a single blood sample for lactate determination at a given submaximal exercise intensity to evaluate endurance fitness for a specific form of exercise. A recent study of soldiers has demonstrated that submaximal exercise tests using lactate concentration at a single exercise intensity are as powerful or more powerful than exhaustive tests with regard to predicting 3000m running time (Jacobs et al. 1985b).

In light of the ease and accuracy of lactate determinations, a strong case can now be made for substituting lactate-related tests for other submaximal or maximal exercise tests without a loss of predictive power. The ability to substitute a submaximal for a maximal test is obviously advantageous because both motivation of the subject and medical precautions are not stringent prerequisites. This may be of particular interest to organisations, e.g. the military, who are often required to perform mass endurance testing for recruitment purposes (Ahlborg et al. 1973).

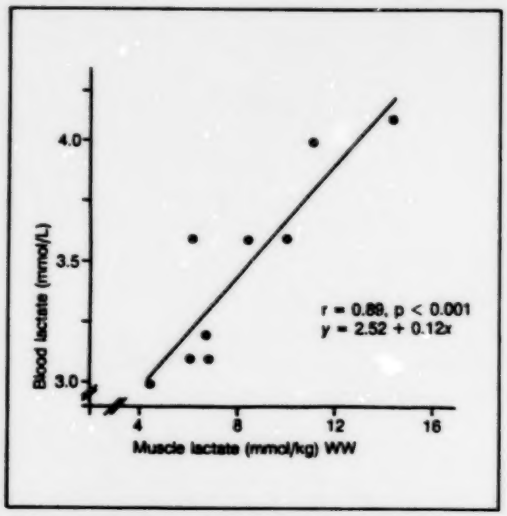

Fig 2. The relationship of blood lactate to muscle lactate concentrations at an exercise intensity closest to that which elicits 4 mmol/L blood lactate during stepwise, incremental cycle exercise. [Reproduced with permission from Jacobs & Kaiser 1982.]

2.3 Reflection of Muscle Fibre Characteristics by Lactate

The question arises as to why lactate circulating in the blood during submaximal exercise should be such a powerful predictor of endurance performance in athletes. Lactate is produced predominantly in the active muscle cell, released to the blood, and a large portion is transported to the liver, heart, and the active musculature where it is consumed. The measured sample is simply the residual net result of all of these processes. Yet a study in which simultaneous blood and muscle tissue samples were taken from subjects at each stage of a progressive exercise test showed that the blood lactate concentrations of about 4 mmol/L are indeed reflective of lactate accumulated within the active muscle (fig. 2). This relationship was not observed at higher or lower blood lactate concentrations (Jacobs & Kaiser 1982). It is tempting to speculate that a specific muscle lactate concentration and the fatiguing effects of the accompanying acidosis (Hultman & Sahlin 1981; Tesch 1980) may act as a threshold for a number of cellular processes at approximately the blood concentrations shown in figure 2.

In addition, the lactate variables discussed above are correlated with several important characteristics of the active musculature: the percentage of slow twitch fibres (Farrell et al. 1979; Sjödin & Jacobs 1981; Tesch et al. 1981), capillary density (Jacobs et al. 1983a; Sjödin & Jacobs, 1981; Tesch et al. 1981), respiratory capacity (Ivy et al. 1980), and the activities of key oxidative and glycolytic enzymes (Sjödin et al. 1981, 1982). The strong relationship between endurance performance and lactate-related variables can probably be attributed to this reflection by lactate during exercise of not only the functional capacity of the central circulatory apparati to transport oxygen to the exercising muscles, but also the peripheral capacity of the musculature to utilise this oxygen, as indicated by some of the metabolic characteristics listed above.

2.4 Endurance Training

Endurance training and/or detraining studies typically employ changes in $\dot{V}O_{2\,max}$ as an indicator of changes in the capacity of endurance exercise. However, it is being increasingly suggested that parameters measured during submaximal exercise may be more valid indicators of training status than $\dot{V}O_{2\,max}$ (Daniels et al. 1978; Katch et al. 1978; Mader et al. 1976). The lactate response to a given exercise is such an indicator and is being increasingly used as a longitudinal marker of the adaptation to endurance training.

A specific criticism of $\dot{V}O_{2\,max}$ centres around the inverse relationship between initial $\dot{V}O_{2\,max}$ and the magnitude of change induced by training (Saltin et al. 1968). It is very difficult to elicit significant increases in $\dot{V}O_{2\,max}$ in highly conditioned athletes in spite of significant improvements in endurance performance (Daniels et al. 1978). In contrast, the results of several studies suggest that lactate-related variables may be more sensitive indicators of training adaptations than $\dot{V}O_{2\,max}$. Williams et al. (1967) first emphasised the dissociation between the relative change in the intensity

Table IV. Comparison of the change in $\dot{V}O_{2max}$ with the change in the blood lactate response to submaximal exercise reported in endurance training studies

Reference	Subjects	Change (%) in $\dot{V}O_{2max}$[a]	Change (%) in lactate variable [a]	Training duration (weeks)
Williams et al. (1967)	Labourers	7	16	4-16
Sjödin et al. (1982)	Elite runners	NS	5	14
Denis et al. (1982)	Students	NS	18	40
Yoshida et al. (1982a)	Students	14	37	8
Denis et al. (1984)	Middle-aged men	8	38	20
Denis et al. (1984)	Students	19	42	20
Hurley et al. (1984)	Young men	26	39	12

a All the percentage changes are statistically significant unless indicated as NS.

at which 'excess lactate' began to accumulate and the relative change in $\dot{V}O_{2\,max}$ (+ 16% *versus* + 7%, respectively) after endurance training; several more recent studies confirm this observation (table IV).

2.5 Optimal Lactate Concentration for Training

Wells et al. (1957) suggested that lactate concentration during steady-state exercise could be used to standardise work intensity. They developed 3 classifications of intensity which were direct functions of lactate concentration expressed as a multiple of normal resting concentrations: (a) light work causing no increase in lactate; (b) heavy work causing 1.5- to 2-fold increases, and (c) severe work causing greater than 5-fold increases in lactate.

A similar classification scheme has been used to quantify training intensity. Several German groups have suggested that the exercise intensity which induces an optimal qualitative stimulus for adaptation to endurance training should elicit a steady-state lactate concentration of approximately 4 mmol/L (Hollmann et al. 1981b; Kindermann et al. 1979; Mader 1980; Mader et al. 1976). This optimal lactate concentration may differ somewhat between individuals and Stegmann et al. (1981) have reported that the individual anaerobic threshold can range from lactate concentrations of 2.0 to 7.5 mmol/L. They have defined this threshold as the highest exercise intensity that can be maintained without a progressive increase in lactate concentration during 20 to 30 minutes of exercise (Stegmann & Kindermann 1982; Stegmann et al. 1981).

Coaches and athletes often believe that the extent of adaptation to endurance training is directly related to the intensity of training. The concept of an 'optimal' training intensity, however, implies that deviations either above or below this intensity result in a reduced training effect. Endurance training at too high an intensity would thus impair the rate of attaining the desired training effect. This contention is supported in a study where the lactate levels of middle and long distance runners were monitored while they trained on a treadmill at a velocity calculated to elicit 4 mmol/L lactate. Those runners who were able to remain closest to the 4 mmol/L concentration during this training run exhibited the greatest increases in the running velocity at 4 mmol/L lactate after the training period. Interestingly, this type of training in these endurance-trained individuals did not change $\dot{V}O_{2\,max}$ but did significantly reduce the ratio of key gly-

colytic to oxidative enzyme activities (Sjödin et al. 1982).

Prescribing training intensity as a function of the lactate concentration elicited by the training may prove to be a means of obtaining a more homogeneous adaptation to training in a group of athletes or subjects than is obtained by setting intensity as a function of maximal heart rate or percentage of $\dot{V}O_{2\,max}$. Such a direct comparison of the effectiveness of various means of establishing training intensity has yet to be empirically examined. However, Hollmann et al. (1981) have presented data showing that a greater endurance training effect was obtained in subjects training at the 4 mmol/L lactate level compared with subjects who trained at 95% $\dot{V}O_{2\,max}$, which probably elicits a much higher lactate concentration.

3. Supramaximal Exercise

The highest lactate concentrations after single bouts of maximal exercise are induced by exercise corresponding in duration to a 400m or 800m run, i.e. lasting for approximately 45 seconds to 2 minutes (Kindermann & Keul 1977). Such lactate levels reach 25 mmol/L, or 15 to 20 times normal resting concentrations. The relevance of such lactate formation to this kind of exercise has both beneficial and maleficent aspects. The maleficent aspect is associated with the established relationship between lactate and both objective and subjective muscular fatigue during short term, high intensity exercise (Noble et al. 1983; Tesch 1980). The beneficial aspect of lactate production concerns the extremely high rate of energy derivation associated with glycolysis or glycogenolysis. The degradation of 1g of carbohydrate to lactate does not yield as much energy as the combustion of 1g of carbohydrate or fat combusted to water and carbon dioxide; however, the rate at which the energy is produced surpasses by far the speed with which energy can be derived aerobically (Brooks & Fahey 1984). This is aptly demonstrated by the high intramuscular concentrations of lactate which are observed after only a few seconds of supramaximal exercise (Hultman & Sjöholm 1983; Jacobs et al. 1982, 1983b; Tesch 1980), an intensity which demands more energy than can be provided via aerobic metabolism. When these concentrations are expressed as a change in lactate per unit time, the rate of change is as high or higher than that which occurs with exhaustive exercise of any other duration (Karlsson 1971; Sahlin 1978; Tesch 1980).

Until recently it was accepted that glycogenolysis leading to lactate formation contributed to energy demands only after the intramuscular stores of energy-rich phosphagens were depleted to a critical level (Karlsson 1971; Margaria et al. 1969). It is now accepted that muscle lactate production and accumulation will occur almost immediately with the onset of exercise which demands more energy than can be provided aerobically, i.e. supramaximal exercise (Jacobs et al. 1983; Sjöholm & Hultman, 1983). Whereas it was previously thought that energy derived from this metabolic pathway would contribute to maximal performance only after 10 seconds of exercise, this new knowledge should be taken into account when developing training programmes for maximal exercise of a few seconds duration. To demonstrate the relevance to an event as short as running a 100m sprint, Hermansen (1971) calculated that the rate of lactate production was maximal after exercise of 10.6 second duration.

3.1 Application of Lactate Analysis to Supramaximal Exercise

A blood lactate evaluation association with such supramaximal exercise could include samples being obtained 1 minute after exercise, e.g. the Wingate or Thorstensson tests described by Inbar et al. (1981), and continually each minute up to the fourth, fifth and sixth minute. Such a test has been administered prior to and after a 6-week sprint training period to examine the kinetics of lactate accumulation following the test, in addition to the actual performance (author's unpublished observations). Adaptive responses to training with this type of exercise include an enhanced ability to 'pump out' lactate quickly from the muscle to the circulation. This is reflected in a smaller difference

after training between the lactate concentration measured 1 minute and 4 minutes after exercise (fig. 3).

Peak lactate concentration after supramaximal exercise of 1 minute duration is often used as an indicator of the metabolic capacity to derive energy from anaerobic pathways. Positive correlations have been reported between running velocities during 100 to 400m sprints and peak lactate concentrations after the run (Fujitsuka et al. 1982; Ohkuwa et al. 1984), although the relationship for competitive sprinters is tenuous (Ohkuwa et al. 1984). Fujitsuka et al. (1982) also demonstrated that this peak lactate concentration could be accurately predicted based on a single blood sample taken 7.65 minutes after exercise – a statistically derived time of appearance of the peak value.

Peak blood lactate values after exhaustive exercise are reliable if activity following exercise is standardised (Fujitsuka et al. 1982). Therefore one possible means of evaluating the competitive performance of exhaustive high intensity exercise would be to sample blood for lactate after competition. If the measured peak values are lower than what is routinely achieved during the best training runs, one would conclude that the athlete was not sufficiently motivated, or could not exert him/herself maximally for some physiological reason.

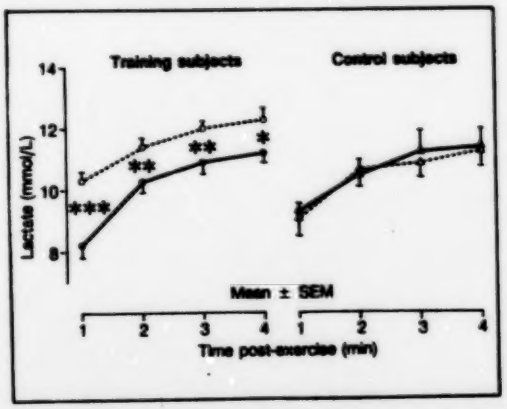

Fig 3. Blood lactate concentration after performance of the 30s Wingate Test. Mean values are shown for the subjects who performed 6 weeks of sprint cycle training (n = 11) pretraining (●——●) post-training (○——○) and for the control group (n = 6) pre (▲——▲) post (△——△) who repeated the test after a similar interval; SEM = standard error of the mean.

4. Recovery and Interval Training

4.1 Performance with Elevated Lactate

Training for competitive sports activities frequently involves interval training. Similarly, actual competition may involve repeated work-rest cycles, such as occurs during ice hockey. Blood lactate concentration during a hockey game rises very rapidly during the players' first playing shift and remains elevated throughout the entire game. This is illustrated in figure 4, which shows the lactate concentration after each playing shift in a forward and defence man during a regular national league game in Sweden.

The results of several studies suggest that high intensity exercise performance of a few seconds (Tesch & Wright 1983; Yates et al. 1983) or a few minutes in duration (Hogan & Welch 1984; Karlsson et al. 1975; Klausen et al. 1972) is impaired if blood lactate levels are elevated prior to exercise, as they are for the hockey players described in figure 4. This is apparently the case even if the exercise is performed by a muscle group other than that which is responsible for the initial lactate production and accumulation (Karlsson et al. 1975; Yates et al. 1983). These impairments may be negligible if the recovery interval is of 20 minutes duration, or the lactate levels do not exceed 4 to 6 mmol/L when exercise is resumed (Weltman & Regan 1983; Weltman et al. 1977).

Tesch and Wright (1983) showed that the lactate concentration after repeated isokinetic maximum voluntary contractions was inversely related to the impairment of peak torque generation when exercise was resumed 40 seconds after the first set of contractions. Their results demonstrate that the rate at which lactate leaves the muscle cell will influence the rate of recovery from local muscular fatigue when supramaximal exercise is subsequently resumed.

Hogan and Welch (1984) were able to isolate the effects of circulating lactate from other possible effects of prior exercise. In their study, subjects

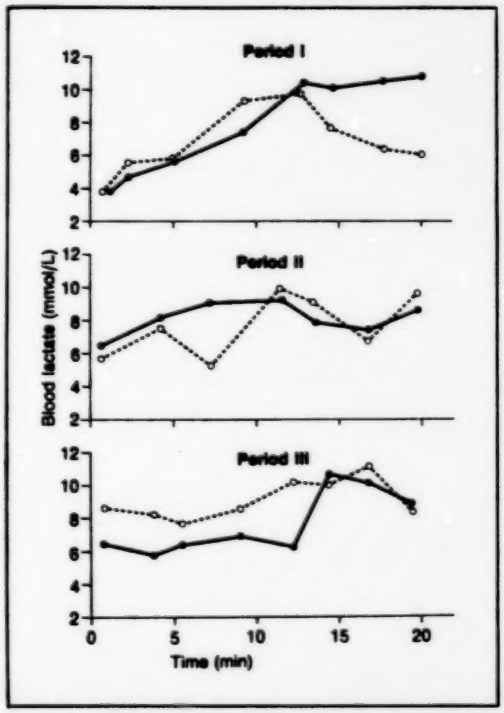

Fig 4. Blood lactate concentration after each playing shift during a competitive ice hockey game for both a forward (●—●) and a defence man (○—○).

performed equal amounts of absolute work on 3 separate occasions during 5 minutes of work at 90% $\dot{V}O_{2\,max}$. A different concentration of oxygen was inspired on each occasion during this initial bout of exercise. After 4 minutes of rest the subjects then exercised to exhaustion at 90% $\dot{V}O_{2\,max}$ while breathing normoxic air. Endurance time decreased as the pre-exhaustive exercise lactate concentration was increased. Such studies have firmly established the benefit of reducing circulating lactate concentrations as rapidly as possible during the recovery interval in a training or competition scenario.

4.2 Optimal Recovery Exercise Intensity

Since Jervell's (1929) initial observation, numerous studies have demonstrated that active recovery increases the rate of lactate removal from the circulation (Davies et al. 1970; Gisolfi et al. 1966; Hermansen & Stensvold 1972; Newman et al. 1937). The identification of an optimal intensity of recovery exercise in this regard has also been the subject of much research. Conflicting results have resulted in supposedly optimal recovery exercise intensities ranging from 22-63% $\dot{V}O_{2\,max}$ (Belcastro & Bonen 1975; Hermansen & Stensvold 1972). This wide range can be partially attributed to the type of ergometer used and the size of the muscle mass recruited during recovery (cycle *versus* treadmill) in the various studies. Hermansen and Stensvold (1972) suggest that the optimal intensity for lactate removal during recovery corresponded to the lowest intensity that elicited an increase above resting lactate values during steady-state exercise, i.e. 60 to 80% $\dot{V}O_{2\,max}$ during treadmill ergometry. Since the relative intensity at which this threshold occurs is directly related to endurance fitness it should follow that the optimal recovery intensity would also be positively related to endurance fitness as suggested by Bonen and Belcastro (1976).

The conflicting findings concerning optimal recovery intensity are therefore not surprising since the subjects in the various studies differed widely in their aerobic fitness. A well-designed study has yet to carefully examine the relevance of aerobic fitness to rate of lactate removal from the circulation during active recovery. Another consideration for establishing recovery exercise intensity is the suggestion that a less variable lactate removal response is obtained in a group if the recovery intensity is expressed relative to the anaerobic threshold, in contrast to expressing it as a percentage of $\dot{V}O_{2\,max}$ (McLellan & Skinner 1982; Stamford et al. 1981).

4.3 Application of Lactate Analysis to Interval Training

Although active recovery is practised by many athletes during interval training, its use during actual competition is minimal, e.g. ice hockey (fig. 4). Between shifts, players remain passive within

the confines of their bench area until their next shift. The empirical evidence cited above (section 4.2) demonstrates clearly that increasing the rate of lactate removed between shifts would enhance the physiological potential for the players to perform to their capacity.

The advent of rapid automated lactate analysers seems applicable to this particular situation. An attempt should be made to examine the effects of designing recovery activities for hockey players that can be performed between shifts, given the physical constraints of the bench. Rapid lactate analysis can also be applied directly to any type of interval training. Coaches in sports such as track, swimming, kayaking, and rowing are routinely monitoring lactate levels after training intervals (personal communications from A. Mader, B. Sjödin, P. Tesch). Heart rate has been traditionally used to signal the time to begin another exercise interval. However, in light of the above findings, it is tempting to speculate that blood lactate may be a better indicator of the readiness of the musculature to receive adaptive stimuli from another bout of exercise.

5. Non-Athletic Applications

5.1 Pathologies

Abnormalities associated with blood lactate accumulation during exercise have proved to be a useful tool in the diagnosis of pathologies. The most easily identifiable pathological response is an inability to derive energy from anaerobic glycogenolysis or glycolysis during exercise. The resultant lack of intramuscular production and accumulation in blood can theoretically be due to an enzymatic impairment at any step in the glycogenolytic pathway (Edwards & Jones 1983). Phosphorylase or phosphofructokinase deficiencies are the usual sources of such rare muscular pathologies (McArdle 1951; Tarui et al. 1965). Lactate accumulation after exercise has also been used in the evaluation of chronic airways obstruction (Cooke et al. 1983) and chronic renal failure (Nakao et al. 1982).

The onset of angina pain during exercise coincides with the intensity at which lactate begins to accumulate during progressive exercise in patients with established coronary artery disease and angina pectoris (Åström et al. 1983). Therefore, it has been recommended that lactate analysis be added to the routine measurements of heart rate, blood pressure and monitoring of heart rhythm usually performed on such patients during stress testing (Karlsson et al. 1983a). A lactate variable associated with a given exercise intensity is probably a more suitable indicator of exercise capacity than is heart rate or percentage of $\dot{V}O_{2\,max}$ for such patients (Coyle et al. 1983). The strong correlation between lactate and perceived exertion (Noble et al. 1983) should further facilitate the prescription of exercise intensities for this and healthy subject populations.

5.2 Exercise and β-Blockade

The blood lactate response to exercise has been used to evaluate the effects of various pharmacological interventions. In particular, the effects of β-adrenergic blockers in this regard have received much attention (for recent review see Kaiser 1984; Tesch 1985). Although intramuscular lactate accumulation after exhaustive exercise is apparently not affected by either a selective or non-selective blocker (Kaiser 1984), blood lactate accumulation is significantly reduced (Kaiser 1984; Schnabel et al. 1983, 1984). There is no difference between the selective and non-selective blockers in this regard. During submaximal exercise the picture is less clear with reports of increased blood lactate at intensities above the 4 mmol/L lactate level with either β_1-selective or non-selective blockade (Kaiser & Tesch, 1983), and decreased concentrations at lower exercise intensities after non-selective blockade (Kaiser et al. 1985). These findings should be considered when comparing the exercise lactate responses between medicated and non-medicated subjects.

6. Limitations and Considerations

As with many other physiological measurements, lactate measurements should be considered

within the framework of the circumstances surrounding the actual measurement. Several factors can influence the rate of lactate production, release and uptake, and thus the final concentration in blood. Blood concentration is a function of turnover and blood flow, and changes in either of these parameters will affect concentration proportionately. A detailed discussion of these parameters is available elsewhere (Donovan & Brooks, 1983).

6.1 Environmental and Experimental Manipulations

At rest and/or during submaximal exercise, lactate concentration can be elevated in the heat (Fink et al. 1975), in the cold (Jacobs et al. 1985a), and may be reduced when the subject is dehydrated (Saltin 1964). Neither heat nor thermal dehydration seem to affect peak blood lactate concentration after supramaximal exercise (Jacobs 1980). If cold exposure is of sufficient duration to cause a decrease in core and/or muscle temperature, lactate values after exhaustive exercise may peak later and be reduced compared to normothermic conditions (Bergh & Ekblom 1979; Blomstrand et al. 1984).

Extreme deviations from normal acid-base balance will also affect the interpretation of lactate variables. Induced alkalosis prior to exercise can cause a higher lactate concentration at a given exercise intensity while the opposite is the case with induced acidosis (Jones et al. 1977; Kowalchuk et al. 1984). It is unlikely, however, that most subjects will evidence the extreme changes in resting pH required to cause such effects. An exception of which one should be wary is the increasing popularity, among athletes involved in supramaximal exercise, of ingesting bicarbonate (Gledhill 1984).

Depletion of muscle glycogen stores either by dietary or exercise manipulations reduces intramuscular lactate accumulation after short term, high intensity exercise (Jacobs 1981). Blood concentrations at specific absolute and relative exercise intensities are also reduced after both submaximal and supramaximal exercise with severely depleted glycogen stores (Jacobs et al. 1981; Jansson 1980; Maughan & Poole 1981; see Yoshida pp 4-9 this issue). This effect persists for several days after extremely prolonged, intense exercise, e.g. a marathon run, even with the resumption of normal dietary routines (Jacobs et al. 1981). Acute consumption of large amounts of glucose may elevate lactate during submaximal exercise while fat consumption can have the opposite effect (Ivy et al. 1981). Abnormally high carbohydrate consumption for several days may cause elevated blood lactate accumulations after both submaximal (Jansson 1980; Yoshida 1984b) and supramaximal exercise (Maughan & Poole 1981). It should be noted however that intramuscular lactate accumulation is not changed after supramaximal exercise in a muscle with glycogen concentrations which are 2 to 3 times normal levels (Jacobs 1981).

6.2 Children and Females

It has been suggested that the capacity for lactate production in prepupertal children is less than in adults because of reduced maximal activities of key glycolytic enzymes such as phosphofructokinase (PFK) [Eriksson et al. 1974; Eriksson & Saltin 1974]. Cumming et al. (1980), however, have taken issue with this contention and demonstrated that peak blood lactate values similar to adult values can be attained by children with sufficient motivation during the exercise test.

It has also been suggested that females possess skeletal muscle characteristics which favour oxidative metabolism and that their capacity to derive energy from anaerobic glycogenolysis is limited compared with males (Komi & Karlsson 1978; Nygaard 1981). An examination of peak lactate values attained by competitive female middle distance runners demonstrates that there is no difference between the sexes in this regard (Mader et al. 1979). A similar finding was reported by Jacobs et al. (1983b) who compared intramuscular lactate accumulation in male and female subjects after performance of the 30-second Wingate Test. The effects of training on glycogenolytic energy production and the ability to tolerate the accompanying perceived fatigue associated with high lactate accu-

Table V. Comparison of reproducibility of $\dot{V}O_{2max}$ and of the power output which was calculated to elicit 4 mmol/L lactate during incremental cycle ergometry. 14 subjects peformed both tests twice within 1 week

	$\dot{V}O_{2max}$ (L/min)		Power at 4 mmol/L (W)	
Test	I	II	I	II
Mean	3.627	3.640	184.1	184.9
SD	0.698	0.741	47.39	52.02
SE	0.187	0.198	12.664	13.90
Range	1.910-4.410	1.830-4.429	77-270	69-274
Method error[a]	0.106		7.125	
Variation[a]	2.9%		3.86%	

a Method error and variation were calculated according to Sale and Norman (1982).
Abbreviations: SD = standard deviation; SE: standard error.

mulation is probably an acquired rather than a sexually mediated 'talent'.

6.3 Procedures Affecting Lactate Measurement

The site of sampling can also influence the time course of lactate accumulation in blood, i.e. arterial *versus* venous *versus* arterialised blood (Yeh et al. 1983; Yoshida et al. 1982b). When the site has been standardised the reproducibility of lactate concentration at given exercise intensities is quite satisfactory (Sjödin et al. 1982) and does not differ significantly from the reproducibility of $\dot{V}O_{2\,max}$. The reproducibility of $\dot{V}O_{2\,max}$ and the exercise power output at 4 mmol/L lactate during incremental exercise is shown in table V. These data were collected from 14 subjects who performed the repeat tests 2 days after the first tests. A more detailed discussion of this topic is found in Graham (1984).

Interlaboratory comparisons of exercise intensities at specific lactate concentrations should be interpreted with care. Since time is required for lactate to be released from the muscle to circulation, the rate at which intensity increases during continuous, incremental ergometry will affect the calculation of an exercise intensity corresponding to a specific lactate accumulation (McLellan 1985; Yoshida 1984a). Some laboratories use rapid incremental tests, increasing intensity each minute, while others emply longer durations at each intensity. Figure 5 demonstrates that a marked difference would result in the calculation of the power output at 4 mmol/L depending on the rate of the increments during exercise.

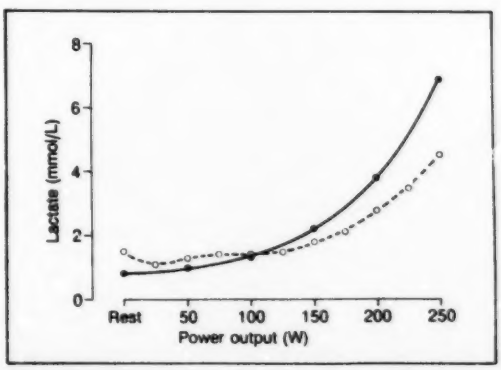

Fig 5. The effect of duration at each state of a continuous, incremental test on blood lactate concentration during cycle exercise at 25 W/min (○----○) and 50 W/4min (●—●).

Changes in the frequency of pedalling during cycle ergometry can also change lactate concentration at a given oxygen uptake (Böning et al. 1984; Löllgen et al. 1980). This is of particular relevance to determining the power output at 4 mmol/L lactate concentration for competitive cyclists. When allowed to pedal at their preferred rate of 90 to 120 revolutions per minute the 4 mmol/L lactate level occurs close to the point of exhaustion if it is attained at all (author's unpublished observations, 1985; P. Tesch, personal communication, 1984). Although intramuscular lactate production may be high in these cyclists, an attractive explanation of the low blood levels is the contention of Donovan and Brooks (1983) that clearance of lactate from the musculature is extremely high for these athletes at the power outputs in question.

The type of ergometer used also influences the intensity at which a given lactate concentration occurs. As is the case with the evaluation of other exercise-induced responses, training specificity should dictate the choice of ergometers, particularly with athletes. With regard to treadmill and cycle ergometers, a lactate concentration of 4 mmol/L will occur at a higher $\dot{V}O_2$ during treadmill exercise in most populations (Hermansen & Saltin 1969; Kindermann et al. 1980). This difference is often attributed to the larger muscle mass recruited during treadmill exercise. However the difference in lactate accumulation described above is not a direct function of the approximately 10% smaller $\dot{V}O_{2\,max}$ typically measured during cycle exercise. Jacobs and Sjödin (1985) observed a 16% greater $\dot{V}O_2$ at 4 mmol/L lactate during treadmill exercise, but only a 9% greater $\dot{V}O_{2\,max}$. Thus the 4 mmol/L lactate concentration occurred at 85% $\dot{V}O_{2\,max}$ on the treadmill but only 79% $\dot{V}O_{2\,max}$ on the cycle – a significant difference. Tesch and Lindeberg (1984) compared lactate accumulation during progressive arm ergometry in elite kayakers, weight/power lifters, body builders and non-athletes and demonstrated that factors other than muscle mass *per se* are responsible for the blood lactate response to exercise.

Repeated lactate-related tests cannot be readily performed within a single test session unless resting lactate levels can be restored prior to the repeat test. Elevated levels prior to exercise will confuse the interpretation of lactate accumulation during subsequent exercise (Davis & Gass 1981).

7. *Conclusion*

This brief description of the uses of blood lactate accumulation in the evaluation of responses to exercise is by no means exhaustive. The rapidity with which related publications are appearing in the exercise science literature makes such a task extremely difficult, yet attests to the growing acceptance of lactate as a valuable diagnostic and interpretative tool. By collating the information presented in this article, it is this author's intention to stimulate further research to clarify the relevance and validity of using lactate in the ways described as well as stimulating applications which will prove to be a value to the practitioner.

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